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LETTER TO THE EDITOR

Clinical-scientific notes

Autoimmune hepatitis triggered by COVID-19

Autoimmune hepatitis (AIH) is an inflammatory liver disease caused by environmental triggers in genetically predisposed patients. Development of acute AIH has been described in patients recently infected with viruses such as Epstein–Barr virus (EBV).[1] We describe a possible case of acute AIH in a patient recently infected with the novel coronavirus disease 2019 (COVID-19).

A 54-year-old man with history of rheumatoid arthritis (RA) and scleritis presented with abdominal pain and jaundice. His RA and scleritis had been treated with infliximab (500 mg/kg every 8 weeks) for the past 10 years. He denied history of liver disease and consumption of alcohol, drugs including antibiotics or supplements.

One month prior, the patient developed fevers and tested positive for COVID-19. He had spontaneous improvement in his symptoms. At the time of his COVID-19 diagnosis, serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were elevated to 233 IU/L and 200 IU/L. Liver function tests (LFT) were previously normal. One month after his diagnosis, he presented with abdominal pain and pruritus. His laboratory tests showed elevated ALT to 1238 IU/L, AST to 1084 IU/L, total bilirubin to 25 mg/dL, alkaline phosphatase (ALP) to 251 IU/L and the international normalised ratio was 1.0.

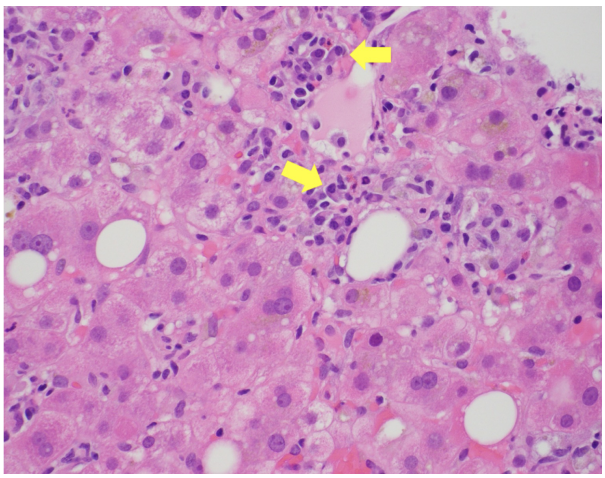

Figure 1 Perivenular as well as lobular inflammation with predominance of plasma cells (arrows). Haematoxylin and eosin staining: ×400.

Serologies for hepatitis A, B, C, E and EBV were negative. COVID-19 polymerase chain reaction was negative. He had an anti-nuclear antibody titre of 1:2560 and an anti-smooth muscle antibody titre of 45 (normal range <20). Previously normal immunoglobulin G (IgG) subclass level was now elevated to 3151 mg/dL (normal range 700–1600 mg/dL). Calculated AIH score was 8.[2] Abdominal ultrasound showed hepatic steatosis. A liver biopsy showed moderate portal inflammation and mild interface hepatitis and moderate lobular inflammation. No fibrosis was seen on trichrome stain (Fig. 1). Given findings concerning for AIH, prednisone (40 mg/day) was started. Infliximab was switched to rituximab for his RA and scleritis. Follow-up laboratory tests 2 months after admission showed significant improvement in his results with total bilirubin, ALT, AST and ALP in the normal range on prednisone (5 mg/day).

Although the most common presenting symptoms of COVID-19 are respiratory, liver injury has been described in as many as 50% of cases.[3–5] LFT are often transiently and minimally elevated but can rise in the setting of sepsis, ischaemia and drug-induced liver injury.[5] Although liver injury is a common finding among patients with COVID-19 infection, the present case was inconsistent with previously described presentations.

Diagnosis of infliximab-induced AIH was also considered. Immune-modulators such as infliximab have been implicated in AIH. It is a rare side-effect, with case reports describing its onset usually within months of starting the drug and up to 2 years.[6] Given the timing of the event coinciding shortly after his COVID-19 infection and his long-standing use of infliximab with no recent changes in dosing, frequency or formulation, infliximab-induced AIH was considered to be lower on the differential. Histological findings had features consistent with AIH; however, this can be largely indistinguishable from viral hepatitis.[2]

Our patient presented with significant jaundice from hepatocellular injury and elevation of IgG 1 month after COVID-19 infection. After starting prednisone and stopping infliximab, the patient's LFT have improved dramatically with resolution of his symptoms. Although an infectious trigger of AIH is not a novel entity, this case illustrates a possible case of COVID-19-induced AIH. Clinicians should be vigilant and aware that COVID-19 can be a trigger of severe AIH even after resolution of infection.

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